chronic obstructive pulmonary disease mortality. More work is needed on the possible nonmalignant effects of workplace exposure to diesel exhaust.

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Health Hazards of Radon Exposure

RADON, A CAUSE of lung cancer in miners of uranium and other underground minerals, has become recognized as a potentially important cause of lung cancer in the general population. This naturally occurring gaseous member of the uranium 238 decay series decays into a series of solid, short-lived isotopes referred to as radon daughters, progeny, or decay products. The release of α-particles by inhaled radon decay products is presumed to damage cells of the bronchial epithelium and thereby cause lung cancer.

As data on indoor air quality have accumulated, it has become apparent that radon and its decay products are present in indoor environments and at unacceptably high concentrations in some homes and other structures. Because the uranium-decay series is present in virtually all rocks and soils, radon contaminates the soil gas that passes into indoor and outdoor air. Thus in homes, the principal source of radon is the soil beneath the home, but building materials, water used in the home, and utility natural gas may also contribute. Measurements made in the United States indicate that the distribution of radon levels in houses is skewed, with the average at about 1.5 pCi per liter, but with many homes in the distribution's tail having much higher levels.

From animal and epidemiologic data, we have a sound understanding of some aspects of radon carcinogenesis. The lung cancer risk has been shown to increase with increasing exposure; the preponderance of the epidemiologic evidence indicates a synergism between cigarette smoking and radon exposure. The epidemiologic studies of underground miners provide the data needed to project the lung cancer risk of indoor exposure to radon. Computer models of the dosimetry of radon decay products in the lungs indicate a comparable potency of radon as a carcinogen with exposures in homes and in mines.

Although substantial uncertainty remains concerning the lung cancer risk associated with indoor radon, all projections indicate that the problem is substantial. Because carcinogenesis by radon is considered to follow a no-threshold exposure-response relation, any exposure, even the average for the population, conveys some risk. Remarkably high risks of lung cancer are projected for high exposures. For the US population, estimates of the annual number of lung cancer cases attributable to radon range from about 5,000 to 20,000 cases.

Health care providers should be prepared to advise patients concerning the risks of indoor radon and not dismiss the problem. Following the 1988 advisory of the Environmental Protection Agency (EPA), the measurement of radon in homes should be advocated. We lack other methods for identifying homes with unacceptable concentrations. Longer term measurements, rather than shorter term measurements with a charcoal canister, are preferred under most circumstances. The results of measurements should be interpreted cautiously, but mitigation should be advised for homes with high levels. The EPA's guidelines offer one framework for interpreting measurements, but the highest acceptable level—4 pCi per liter—is not a boundary between safe and unsafe levels. Smokers should be cautioned about the synergism between smoking and radon exposure and advised to stop smoking.

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Male Reproductive Toxicity

WORK-RELATED BIRTH DISORDERS are commonly regarded as related to the occupational experiences of pregnant women. Evidence suggests, however, that toxic exposures in men can also influence reproduction. There are many theoretic mechanisms by which this might occur: a pregnant woman might have direct exposure to a toxin inadvertently transported home from the workplace; a systemic, endocrine, or testicular toxic reaction could impair the male libido or fertility; and systemically absorbed chemicals that cross the blood-testis barrier or are secreted into seminal fluids might either damage sperm or directly affect a fetus or oocyte through sexual transmission.

A number of workplace chemicals have been reported to cause sperm abnormalities. Lead exposure, for example, has been shown in a study of 150 male storage battery workers to be dose-related to the prevalence of oligospermia, sperm hypomotility, and abnormal sperm characteristics. Exposures to boron, cadmium, manganese, and mercury have each been linked to sperm abnormalities, but these data come from small studies or case reports and are inconclusive. Abnormal spermatogenesis has also been described in epidemiologic studies of men with exposure to certain organic compounds, including carbon disulfide, chloroprene, dinitrotoluene and toluene diamine, and the pesticides carbaryl, chlordecone, and dibromochloropropane (DBCP). In many instances these sperm abnormalities have been of indeterminate clinical significance, but human exposures to lead and to DBCP have produced infertility, and the more severe cases of DBCP spermatotoxicity have been irreversible. There is generally minimal information from studies in humans or animals with which to judge the potential spermatotoxicity of the thousands of other commonly used industrial chemicals.

Many authorities question whether paternal chemical exposures can influence a partner's pregnancy outcome or cause abnormalities that are transmissible to offspring, but there is evidence that such adverse effects occur. Paternal exposures to lead, chloroprene, and DBCP have each been reported to increase rates of spontaneous abortion. Other epidemiologic studies have also suggested, for example, that partners of male anesthetists and of copper smelter workers are at risk for spontaneous fetal loss and that paternal "hydrocarbon" exposure may increase the occurrence of low birth weight, central nervous system malformations, and childhood cancers. Although few human studies have been confirmed with subsequent studies, they are supported in principle by the findings of animal research in which exposures of male animals to various alkylating agents have subsequently produced genetic mutations, chromosomal translo-

cations, malformations, and tumors detectable in first- and, in some studies, second-generation offspring.

There is a paucity of data regarding male reproductive toxicity, but the implications of the available data are broad. Workplace exposures should be considered in evaluating male infertility. Men attempting to start a family should be counseled to minimize chemical exposures. Finally, efforts to reduce workplace exposures to known or suspected reproductive toxins should be extended to both men and women.

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Ethylene Oxide Carcinogenicity

ETHYLENE OXIDE (C_2H_4O) is an important industrial chemical used in sterilization and many manufacturing processes. At ambient temperatures it is a gas. In vivo it is rapidly distributed throughout the body. Short- and long-term exposures may lead to respiratory irritation, central nervous system depression, and seizures. At high concentrations ethylene oxide can induce lethal mutations and is embryotoxic in animals. Ethylene oxide is able to alkylate DNA, causing gene mutations that lead to sister chromatid exchange abnormalities and chromosomal damage.

Ethylene oxide has been found carcinogenic in animals following subcutaneous, oral, and inhalation administration. Following oral administration, ethylene oxide produces an increase in local tumors, most notably squamous cell carcinoma of the forestomach in rats. Following inhalation ethylene oxide produces a substantial increase in the incidence of mononuclear cell leukemia in male and female rats and in that of peritoneal mesotheliomas in male rats. An increase in the incidence of brain tumors (rarely found in control animals) is also noted in male rats with exposure to ethylene oxide by inhalation.

Epidemiologic evidence shows an association between the exposure of humans to ethylene oxide and some forms of cancer. An excess of leukemia mortality has been reported in several epidemiologic studies, but these studies do not provide conclusive evidence for this association owing to the small number of workers studied and the possibility of exposure of the workers to other carcinogens. These studies, however, do provide some evidence for ethylene oxide's carcinogenicity in humans.

In 1985 the International Agency for Research on Cancer concluded that there is sufficient evidence for the carcinogenicity of ethylene oxide in animals and that the evidence of carcinogenicity in humans is limited. Overall, the review of this information suggests that the data on both animals and humans is sufficient to classify ethylene oxide as probably carcinogenic in humans.

In light of the importance of sterilizing medical equipment, especially with the danger of human immunodeficiency virus and hepatitis B infections, ethylene oxide will continue to be widely used. Human exposure to ethylene oxide should be kept as low as feasible given present-day technology. Health personnel working in close proximity to ethylene oxide should be given proper training as to ethylene oxide's dangers and be informed as to the known and uncertain risks of ethylene oxide exposure. The function of sterilizing equipment should be regularly assessed, proper ventilation established, and alarm systems installed.

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Occupational Back Pain of Nurses

HEALTH CARE WORKERS are potentially subject to a large number of occupational health hazards, ranging from carcinogens and mutagens such as ethylene oxide and chemotherapeutic agents to the effects of rotating shift assignments. Musculoskeletal disorders are also prominent among the problems faced by health care providers. Occupational back pain among nursing staff is common and leads to a considerable loss of valuable nurses. Epidemiologic studies in several countries—England, United States, Denmark, Israel—provide rather uniform estimates of the high frequency of such illnesses. An analysis of workers' compensation claims suggests that acute care hospital nurses are at risk and that nurses in chronic care facilities are at particularly high risk. For example, registered nurses are 5.1 times as likely to have back-related compensation claims as are cashiers, and nurses' aides are 22 times as likely.

In the past, much effort has been expended in training student nurses in the "proper-lift" technique to be used in patient transfers. There are several reasons why this has not succeeded in controlling the problem: First, the techniques taught may actually increase low-back stress. Second, other nursing activities besides lifting patients—such as moving equipment and standing in awkward positions for prolonged periods—may also be important in producing these problems. Third, such training may account for the attitude of many nurses that they are personally responsible for preventing their own health problems, thereby directing attention away from environmental factors. Fourth, many lifting assistance devices, such as the Hoyer lift, are awkward to use and so are underused. Fifth, hospital staffing patterns may not permit the use of two-person lifts. Sixth, understaffing and changes in work schedules, particularly the use of 12-hour shifts, may be exacerbating musculoskeletal work stresses. Finally, counting on workers to modify their work practices to decrease occupational injury and illness rates is generally less effective than appropriate environmental modifications. Successful intervention programs require a major commitment to providing help for individual nurses rather than providing "training" in groups.

Although back injury rates of nurses approach those of truckers and warehouse workers, nurses have a fundamentally different problem because they may risk injury to themselves in order to preserve and improve the health of their patients. In addition to continuing traditional proper-lift training and providing symptomatic treatment, it is important to encourage the proper reporting of nursing injuries, to encourage nurses to analyze their activities carefully, to apply well-known industrial ergonomic principles to hospital and hospital equipment design, and to consider carefully the